pendix is very thick, and in two places there are obliterations of the lumen; at the tip and near the base, there is also a yellowish diffuse mass in the mucosa near the base. At one place in the wall of the appendix there is a mucocele measuring 1.5 centimeter in diameter.

Microscopic.—Section through the yellowish colored mass in the mucosa shows it to be a carcinoma containing solid groups of cancer cells. Section through the wall of the ruptured cystic cavity mentioned above shows an inflammatory and hemorrhagic connective tissue wall.

Diagnosis.—Chronic appendicitis with appendiceal carcinoma and a mucocele. Apparently the carcinomatous tissue is completely removed. This type of carcinoma exhibits only a small degree of malignancy.

Structure.—Two main varieties of the tumor appear: (1) columnar cell or gelatinous adenocarcinoma, and (2) small polygonal, spheroidal cell alveolar carcinoma. The former type presents the same age incidence (fifty-two years) and general malignancy as other similar intestinal carcinomas, while the former occur at any early age and are almost invariably benign (Rolleston, Jones). Transitional forms of intermediate age incidence are observed.

Carcinoma of appendix has been recognized and emphasized by Elting, Maschkowitz, Batzdoff, McCarthy, Zaaijer, Milner, McWilliams, Kudo, Roketansky, Balduf, Batzdof, Konjetzny, Kelly, and Neugibauer. The clinical course is merged with that of chronic appendicitis (McWilliams, Rolleston, Jones, Lit.), emphasizing the principle that each organ has its own form of carcinoma.

In conclusion, patient made a rapid recovery. So far no untoward symptoms of return.

Patient working every day.

1202 Brockman Building.

JAUNDICE IN ACUTE INFECTIOUS MONONUCLEOSIS (GLANDULAR FEVER)

REPORT ON TWO CASES

By V. R. MASON, M. D.
Los Angeles

A LARGE number of cases of glandular fever have been reported in recent years, and comprehensive summaries of the symptomatology may be found in modern textbooks and collective reviews. Jaundice as a prominent symptom or complication was reported first by Mackey and Wakefield[1] in 1926, and no other similar case has been recorded in the literature up to the present time.

Their patient was a white, male, twenty-three years of age, whose illness began with fever, sore throat, and glandular enlargement. On the sixth day of the illness jaundice was noticed, and the patient complained of dull epigastric pain. The tonsils had been removed previously. The liver and spleen were slightly enlarged and there was moderate general glandular enlargement. The leukocyte count was 15,800 per cubic millimeter, with 91 per cent mononuclears. The Wassermann reaction with serum was negative. No bile salts were present in the feces. No mention was made of bile in the urine. There was an immediate direct van den Bergh reaction with serum. The patient made a complete recovery and at the end of two months the blood counts were normal.

The two cases to be reported here are similar to the case reported by Mackey and Wakefield.

CASE REPORTS

CASE 1.—A white, male, twenty-three years of age, became ill October 14, 1924, with malaise, fever, coryza, nausea, and pain in the "pit of the stomach." There was considerable abdominal distress after eating. When first examined, October 20, 1924, his temperature was 102 degrees F. The superficial lymph nodes were all moderately enlarged. The pharynx was red and the tonsils were large and red. The spleen was felt just below the costal margin. There was considerable tenderness on palpation of the epigastrium, but the liver was not felt. The hemoglobin and red cell count were normal. The leukocyte count was 15,700 per cubic millimeter with 84 per cent mononuclear cells. The fever varied from 101 to 102 degrees F. each day. On October 25, 1924, there was more marked epigastric tenderness and the liver edge was felt at the costal margin. There was also moderate jaundice of the skin and sclerae. The urine contained bile. The feces were of normal color. The jaundice persisted for nearly two weeks. The fever gradually subsided and the patient made an uneventful recovery. January 23, 1925, the leukocyte count was 7300 per cubic millimeter with 37 per cent mononuclear cells and 62 per cent polymorphonuclear neutrophiles.

CASE 2.—G. C. M., a white, male, twenty years of age, was taken ill March 2, 1928, with malaise, chills and fever. He was confined to bed for a week and then attempted to be about, but weakness, fever and night sweats forced him to bed again. His throat became tender and sore. When first examined, March 19, 1928, there was moderate enlargement of the superficial lymph nodes and marked jaundice of sclerae and skin. The tonsils had been removed previously, but there was a dirty, patchy exudate over the pharyngeal mucous membrane. The spleen was palpable just below the costal margin. The liver area was tender on pressure, but the liver was not felt. The urine contained traces of albumin and bile. The feces were of normal color and contained bile. The leukocyte count was 15,400 per cubic millimeter with 87 per cent mononuclear cells. Hemoglobin and red cell count were normal. Platelets were unusually numerous in the smears. There was a positive direct Van den Bergh reaction with serum. The icteric index was 39. A Wassermann reaction with jaundiced serum was doubtful, but later both Kahn and Wassermann tests were negative. The exudate in the pharynx contained many cocci and a few spirallar and fusiform organisms. The fever varied from 99 to 101 degrees F. for a few days, and then gradually receded. March 27, 1928, the leukocyte count was 8000 per cubic millimeter with 74 per cent mononuclears. The patient made an uneventful recovery.

Discussion.—The clinical and hematological features of these three cases are so similar and typical that it seems improbable that an error in diagnosis could have been made. A typical form of acute catarrhal jaundice was readily excluded by study of the blood smears, and acute leukemia was ruled out by the benign course. The blood picture of my cases differed in no particular from that observed in other examples of this disease and reported in detail by Evans and Sprunt,[2] and Downey and McKinlay.[3] Most of the mononuclear cells resembled those present in normal blood and contained many azurophile granules. There were also large numbers of lymphocytes, both large and small forms, some of which were apparently very young. In the cases of glandular fever which I have observed, the mononuclear cell formula of the blood has varied as described by Downey and McKinlay.[3] Histocytes have predominated in some cases and cells of the lymphocytic series in others. This is not remarkable since the organs grossly involved in the disease house the lymphatic and reticulo-endothelial cell systems. Sections of lymph nodes and tonsillar tissue reported on by Evans and Sprunt,[2] Downey and

McKinlay,[3] Fox[4] and Longcope,[5] showed no pathognomonic alterations. The striking alteration, however, was lymphoid hyperplasia with increase of mononuclear cells. It seems reasonable to assume that the blood pictures encountered in this disease might vary from case to case. I have seen outpourings of similar cells in great numbers in estivo-autumnal malaria, hemorrhagic smallpox, and sepsis. Similar "leukemoid" reactions have been reported in other conditions and have led to errors in diagnosis.

The platelets in glandular fever are always present in increased numbers from estimates of the stained smears of patients I have observed.

The cause of the jaundice in the three cases is of considerable interest. Mackey and Wakefield believed it was dependent either upon acute cholecystitis or pressure on the ducts by enlarged glands in Calot's triangle.

The occurrence of jaundice in three similar cases, however, makes those causes unlikely, and it would seem more reasonable to assume that an acute hepatitis with disturbance of the excretory function of the hepatic cells from swelling and inflammation led to a high bilirubin content of the blood stream with visible jaundice.

523 West Sixth Street.

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COCCIDIOIDAL GRANULOMA

CASE REPORT

By PAUL R. WALTERS, M. D.
Dinuba

THE appearance of an unrecognized disease in this community among several of the same nationality, with one death and one patient moribund, and the loss of contact with two others suffering from apparently the same disease justifies the presentation of this brief résumé and report.

Coccidioidal granuloma has been proven from a study of the cases reported, to be a disease peculiar to the San Joaquin Valley in California. occurring in residents or in persons who have been residents. The disease is infrequent. I have lived in Dinuba, San Joaquin County, for the past fifteen years; the case reported is the first to come under my observation.

Definition.—Coccidioidal granuloma may be defined as an infrequent subacute or chronic infectious disease caused by a specific mould fungus, *Coccidioides immitis*. It is characterized clinically by cough, expectoration (frothy in character), elevation of temperature, acceleration of pulse rate, emaciation, weakness, and finally death; locally by glandular enlargements, and areas of edematous, reddened, scattered masses over the body, which either break down or exude a substance like ground liver mixed with purulent fluid when incised.

History.—This rare disease was first described by Wernicke of Buenos Aires in 1892. The second case reported, the first recognized in America, was reported by Rixford and Gilcrist in 1896. Many interesting and valuable observations, studies and investigations have been made since the report of the first case, among which might be mentioned the studies of Ophüls,[1] Philip King Brown,[2] Cummins,[3] and Rixford of San Francisco and the serological work of Hirsch[4] and Benson of New York, and the roentgen studies of Taylor[5] of Los Angeles. The literature contains many interesting papers on this disease.

Geographic Distribution.—As previously stated, a review of the literature brings out the fact that the majority of the reported cases occurred in the San Joaquin Valley in California or in persons who had resided in this valley.

Etiology.—The disease is caused by a specific mould fungus named by Rixford, *Coccidioides immitis*. It belongs to the class *Ascomycetes*. It is a spheroidal double encapsulated body measuring from 20 to 40 microns in diameter, contains spores, and in aërobic culture a separate mycelium is produced. The habitat is unknown; it is supposed to be taken into the body through the respiratory tract by inhalation.

Morbid Anatomy.—The pathology of the disease in the internal organs closely resembles tuberculosis, while the external lesions resemble blastomycotic infection. The disease seems to begin in the respiratory tract, and is spread by the blood and lymphatic system. The bones, lymphatic glands, skin and soft tissue become involved before death.

Symptoms.—The disease presents itself as an ordinary cold or bronchitis, which may clear up temporarily. There seems to be a lull in the respiratory symptoms after about three weeks. The temperature is irregular and ranges from 99 to 101 or 102. The lymphatic glands enlarge, especially those in the cervical region; break down, and leave permanent sinuses. Soon various enlargements occur on the surface of the body like fair-sized furuncles, which are painful. Relief is obtained after drainage is established. These localized infections involve the bone and soft tissue. The patient becomes asthenic, emaciated, and later dies.

Diagnosis.—Race seems to play no part in this disease although the cases that I have seen are all in the Filipino race. Sex: the ratio is about twenty males to one female. Age: most of the cases occur in early or middle life although Reisman reported a case in a patient five years of age. The diagnosis may be established upon finding the causative mould. The absence of the